THE BLOOD-PRESSURE IN PNEUMONIA.

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From a clinical stand-point the mode of death in pneumonia is of the greatest importance. On this determination the prognosis and treatment must necessarily in greater part depend. It is safe to say that the larger number of clinicians will accept the statement of von Jürgenson that, "Patients who die of pneumonia are killed by cardiac insufficiency."

The factors assigned in producing the cardiac failure are (1) the pneumonic exudate interfering with and obstructing the pulmonary circulation, thus increasing the work of the right heart; (2) the pneumonic changes in and about the lung resulting in a disturbance of the normal pulmonary influence in maintaining the circulation and (3) the injurious effects of the fever on the heart.

One of the important problems in the study of pneumonia is that associated with the causative factors of the cardiac failure.

The slight mechanical influence on the lesser circulation and the interference of the respiratory assistance to the circulation can hardly be seriously considered, especially when a small localized consolidation produces a fatal outcome or an enormous, perhaps, complete involvement of one entire lung, goes on to a favorable termination. Even the myocardial degeneration described by von Jürgenson, Zenker, and Liebermeister as due to the pyrexia was brought into question by Aufrecht when he directed attention to cardiac failure in pneumonia with slight fever and the infrequent occurrence of the same in diseases with long-protracted fever, as in typhoid.

However, it must be admitted that pneumonia patients die and the fatal termination is usually manifest by signs and symptoms of circulatory failure. At present it is generally admitted that the circulatory disturbance is produced by the pneumococcal toxemia, but how or in what manner has not been definitely determined.

Admitting that the fatal issue is brought about by a circulatory failure, the result of the toxemia, it is evident that some portion of the cardiovascular apparatus must be involved. For practical purposes this means anatomical or functional changes in either the heart or the vascular system, including the contained blood, while, undoubtedly, in some instances there is a simultaneous involvement of the entire circulatory apparatus.

Regardless of a few exceptions, it was formerly quite generally accepted that the myocardium was at fault, until Romberg[1] and his associates investigated the problem experimentally and arrived at the conclusion that the circulatory failure invariably followed an exhaustion or paralysis of the vasomotor center in the medulla. Since that time up to recently the vasomotor theory has held full sway, to the complete exclusion of the previously accepted idea of myocardial degeneration.

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[1] Experimentelle Untersuchungen über die Allgemeine Pathologie der Kreislaufstorung bei Acuten Infectionskrankheiten, Arch. f. klin. Med., 1899, lxiv, 652.

However, as might be expected, recent experimental and clinical investigation has again emphasized the importance of the cardiac involvement and with a corresponding diminution of the influence of the vasomotor mechanism.

Newburgh and Minot,[2] in their clinical study of pneumonia, among other findings, arrived at the conclusion that the blood-pressure curve did not suggest a failure of the vasomotor center. This conclusion has been amply confirmed experimentally by W. T. Porter and L. H. and I. Newburgh.[3] They were able to demonstrate that the vasomotor center was not impaired in fatal pneumonia.

Even more convincing are the microscopic findings of Willson,[4] as determined in the fatal pneumonias. In every instance he was able to detect myocardial changes due either to a toxemia or a local infection. That it could be otherwise is hardly to be expected, when it is recalled that fatal pneumonia is accompanied by a toxemia and practically every case with a pneumococcal septicemia.

With the evidence at hand it is apparent that no definite and final statement can be made, further than fatal pneumonias die with the clinical manifestations of a circulatory failure, the result of a pneumococcal toxemia and septicemia; but whether the myocardium or the vasomotor apparatus is chiefly involved remains in doubt. Perhaps it may be ultimately determined that there are various groups in which one or the other predominates, while in some, both the myocardium and vasomotor system are concerned.

On the experimental findings of Romberg and his associates,[5] G. A. Gibson[6] formulated a statement in reference to the relation of the blood-pressure to the pulse-rate, known as the Gibson rule, which is as follows: "When the arterial pressure, expressed in millimeters of mercury, does not fall below the pulse-rate, expressed in beats per minute, the fact may be taken as of excellent augury, while the converse is equally true. From the work of the last few years in my own wards no fact is more certain than this."

During the past few months a careful clinical study of the application of this rule has been made of the pneumonias admitted to the Ward Eight Medical Service at the County Hospital. In each instance a careful record was made of the pulse, temperature, and respiration rate every four hours. The systolic blood-pressure, by the auscultatory method, was determined at least every twelve

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[2] The Blood-pressure in Pneumonia, Arch. Int. Med., 1914, xiv, 48.

[3] The State of the Vasomotor Apparatus in Pneumonia, Am. Jour. Physiol., 1914, xxxv.

[4] The Heart in the Pneumonias, Jour. Am. Med. Assn., 1914, lxiii, 981.

[5] Loc. cit.

[6] Some Lessons from the Study of the Arterial Pressure, Edinburgh Med. Jour., 1908, n. s., xxiii, 17.

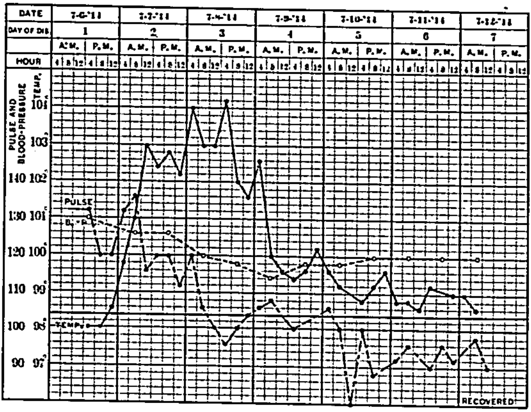

CHART I.—Chart from a case of pneumonia with recovery, showing blood-pressure well above pulse-rate.

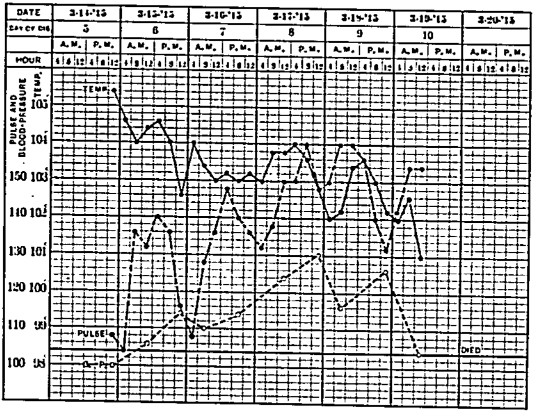

CHART II.—Chart from a fatal case of pneumonia in which blood-pressure fell far below pulse-rate.

hours. From these records a chart was made showing the pulse-rate and blood-pressure curves. For convenience the temperature curve as well as the time and amount of digitalis administered, were also included. The number of pneumonias so charted includes 31 cases, which may be divided, for further study, into two groups according to the relative position of the blood-pressure above or below the pulse-rate.

GROUP 1—BLOOD-PRESSURE CONSTANTLY, OR IN GREATER PART, ABOVE THE PULSE-RATE.

No.	Name.	Age.	Involvement.	Leukocytes.	Day of illness.	Complications.	Result.	Gibson rule.
1	M. M.	39	Left lower	14,000	4	Mitral insufficiency Nephritis	Died	Negative.
2	O. E.	44	Right lower	8,128	4	Mitral insufficiency	Recovered	Positive.
3	D. N.	39	Left lower	...	3	....	Recovered	Positive.
4	M. G.	29	Left lower	19,350	2	....	Recovered	Positive.
5	M. R.	44	Right upper	19,400	4	Mitral insufficiency	Recovered	Positive.
6	M. M.	24	Right lower	14,000	?	....	Recovered	Positive.
7	F. S.	63	Both lower	24,000	7	Myocarditis Nephritis	Died	Negative
8	E. C.	48	Left lower and upper	29,300	6	....	Recovered	Positive.
9	V. C.	27	Right lower	11,700	1	....	Recovered	Positive.
10	T. T.	23	Left lower	14,000	7	....	Recovered	Positive.
11	S. G.	58	Left lower	80,000	3	Myocarditis Obesity	Died	Negative.
12	V. M.	21	Both lower	20,000	4	Pleurisy	Recovered	Positive.
13	A. McD.	48	Right lower and middle	14,000	5	Nephritis	Recovered	Positive.
14	M. M.	56	Left lower (complete) Left upper (partial)	33,800	?	Pleurisy Dilated heart	Died	Negative
15	H. E.	70	Right lower	31,300	6	Nephritis Myocarditis	Died	Negative.
16	M. E.	43	Right lower	17,700	5	Bilateral pleurisy	Recovered	Positive.
17	E. C.	64	Left upper	27,800	6	....	Recovered	Positive.

Summary: Of the 17 cases, 12 complied with the rule, or 70.5 per cent.

GROUP 2—BLOOD-PRESSURE CONSTANTLY, OR IN GREATER PART, BELOW THE PULSE-RATE.

No.	Name.	Age.	Involvement.	Leukocytes.	Day of illness.	Complications.	Result.	Gibson rule.
1	J. K.	38	Right lower and right upper	...	5	....	Recovered	Negative.
2	T. W.	49	Left lower	20,500	7	Nephritis	Died	Positive.
3	L. S.	19	Both lower	15,000	8	Pleurisy	Recovered	Negative.
4	M. L.	61	Both left and right upper	14,250	?	....	Died	Positive.
5	A. M. R.	41	Right upper and middle	26,250	8	....	Died	Positive.
6	L. L.	13	Right upper	37,550	3	....	Recovered	Negative.
7	M. V.	35	Right lower and middle	11,700	9	Nephritis	Died	Positive.
8	N. W.	42	Right lower	16,500	8	....	Died	Positive.
9	E. D.	18	Left lower and right empyema	11,300	5	Empyema	Recovered	Negative.
10	J. S.	56	Left lower	11,800	1	....	Died	Positive.
11	G. C.	22	Left lower	36,000	6	Pleurisy	Recovered	Negative.
12	D. A.	32	Both lower	16,400	6	Pregnant (5 months) Nephritis	Died	Positive.
13	C. B.	26	Right lower	12,600	7	Pleurisy	Recovered	Negative.
14	A. D.	28	Both lower	15,100	5	....	Died	Positive.

Summary: Of the 14 cases, 8 complied with the rule, or 57.1 per cent.

As will be observed in the first group of 17 cases, 12, or 70.5 per cent., complied with the rule, while in the second group of 14 cases, 8, or 57.1 per cent., were positive, thus agreeing with the rule. Of the total of the two groups, 31 cases, 20, or 64.5 per cent., verified the observations of Gibson.

A reasonable explanation for four of the five discrepancies in the first group is to be found in the presence of a nephritis in three and a myocarditis and obesity in the fourth.

In the second group, of the 6 deviations from the rule, 3 were comparatively young, with soft elastic vessels, in which presumably a relatively lower pressure existed before the onset of the pneumonia, or, at least, were able to withstand a greater depression of the circulation.

Excluding the 7 cases in which a reasonable exception existed, of the remaining 24 only 4 failed to comply with the rule.

Aside from the prognostic significance, the blood-pressure and pulse ratio have been of the greatest assistance and satisfaction as a guide to the administration of cardiac stimulants. In some no stimulation was used, while in others one or two intravenous injections of digitalis were sufficient to reduce the pulse-rate, increase the blood-pressure, and produce a crossing of the curves.

REGENERATION OF BONE.[1]

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The regeneration of bone, formerly with reference to the repair of fractures and in the past century to the ultimate fate of the bone transplant, has been a much-debated topic. One of the earliest and foremost investigators in this field of research was the French naturalist Duhamel du Monceau, who, in 1739, called attention to the active role played by the periosteum in the regeneration of bone. But the exhaustive work of the eminent French surgeon Dupuytren, placed the subject upon a scientific basis. Ollier, Barth, Axhausen, and others have presented many valuable contributions to the subject.

Cellular Dynamics. Some idea of cellular dynamics is essential for an adequate understanding of the physiological mechanism of

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[1] This article does not refer to the transplantation of compact bone into cancellous bone.